Seasonal and temperature modifications of the association between fine particulate air pollution and cardiovascular hospitalization in New York state

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Abstract

It is known that extreme temperature and ambient air pollution are each independently associated with human health outcomes. However, findings from the few studies that have examined modified effects by seasons and the interaction between air pollution and temperature on health endpoints are inconsistent. This study examines the effects of short-term $PM_{2.5}$ (particulate matter less than or equal to 2.5 μm in aerodynamic diameter) on hospitalization for cardiovascular diseases (CVDs), its modifications by season and temperature, and whether these effects are heterogeneous across different regions in New York State (NYS). We used daily average temperature and $PM_{2.5}$ concentrations as exposure indicators and performed a time series analysis with a quasi-Poisson model, controlling for possible confounders, such as time-relevant variables and dew point, for CVDs in NYS, 1991–2006. Stratification parametric models were applied to evaluate the modifying effects by seasons and temperature. Across the whole year, a 10-μg/m^3 increment in $PM_{2.5}$ concentration accounted for a 1.37% increase in CVDs (95% confidence interval (CI): 0.90%, 1.84%) in New York City, Long Island & Hudson. The $PM_{2.5}$ effect was strongest in winter, with an additional 2.06% (95% CI: 1.33%, 2.80%) increase in CVDs observed per 10-μg/m^3 increment in $PM_{2.5}$. Temperature modified the $PM_{2.5}$ effects on CVDs, and these modifications by temperature on $PM_{2.5}$ effects on CVDs were found at low temperature days. These associations were heterogeneous across four $PM_{2.5}$ concentration regions. $PM_{2.5}$ was positively associated with CVD hospitalizations. The short-term $PM_{2.5}$ effect varied with season and temperature levels, and stronger effects were observed in winter and at low temperature days.

Graphical abstract

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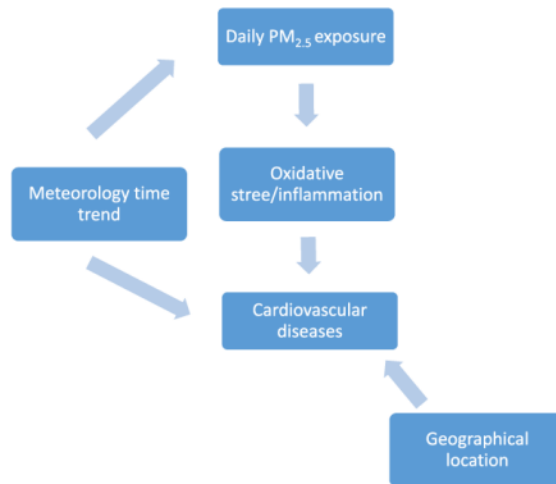
Keywords

CVD; $PM_{2.5}$; Temperature; Season; Modification

1. Introduction

The short-term association between airborne particles and adverse health effects has been documented, with evidence suggesting substantial increased risk of mortality, hospitalization, and emergency department visits (Lin et al., 2012; US EPA, 2004). Temperature can also affect health, and it is well established that extremely high temperatures contribute to increases in mortality and some morbidity, such that a U-, V-, or J-shaped relationship between temperature and health endpoints is usually found (Curriero et al., 2002; Hajat et al., 2006; Kovats et al., 2004). The weather condition is a major driving force of air pollution concentration (Jacob and Winner, 2009; Kinney, 2008; Tai et al., 2010). For example, higher temperature speeds up chemical reactions in the air; lower temperature makes the particulate matter (PM) dissipate in the air more slowly than usual; and rain washes out water-soluble pollutants and PM. Additionally, temperature is associated with season and PM concentration varies with region and season (Bell et al., 2008). $PM_{2.5}$ (PM less than or equal to 2.5 μm in aerodynamic diameter) has been observed to be greatest in winter in some locations. Fares (2013) found that $PM_{2.5}$ increased up to 57% in winter in China and the concentration of $PM_{2.5}$ and PM_{10} (PM less than or equal to 10 μm in aerodynamic diameter) in Turkey was higher in winter than in summer. However, air pollution was also found to be worse during a heatwave in a few studies. These studies describe associations of air pollution with temperature, but the results are not consistent.

Few epidemiological studies have looked at the interaction between PM and temperature and the effect of seasonality, especially on $PM_{2.5}$, and these few studies have produced conflicting results. Bell et al. (2008) reported seasonal variation in the association between $PM_{2.5}$ and cardiovascular hospitalization with the strongest estimated association in winter. However, Katsouyanni et al. (1993) found an interaction effect between high levels of

particulate air pollution and high temperature on mortality. Roberts (2004) and Ren et al. (2006) suggested that it is important to consider the possibility of an interaction between daily particular air pollution and daily mean temperature on mortality. Additionally, Samet et al. (1998) and Hales et al. (2000) found little evidence that weather conditions modified the effect of air pollution on mortality. As the prior results are inconsistent and there is an association of air pollution with temperature, the question of whether temperature modifies the effect of $PM_{2.5}$ on cardiovascular diseases (CVDs) remains to be addressed.

The purpose of this study was: (1) to identify the potential health impact of $PM_{2.5}$ on CVD hospitalizations in New York State (NYS) during the period 1991–2006, while controlling for time-relevant confounding variables; (2) to investigate the seasonality of the potential health impact of $PM_{2.5}$; (3) to evaluate a possible modification by temperature of the effect of $PM_{2.5}$; and (4) to examine whether the above-mentioned effects differ across regions.

2. Materials and methods

2.1. Morbidity data

The target population included all NYS residents with hospital admissions due to CVDs during 1991–2006. A CVD case was defined according to a principal diagnosis with the following International Classification of Disease, 9th Revision, including chronic rheumatic heart disease (ICD-9 codes 393–396), hypertension (401–405), ischemic heart diseases (410–414), cardiac dysrhythmias (427), heart failure (428), and cerebrovascular diseases (430–434, 436–438). Discharge data for hospital admissions due to CVDs among residents of NYS from 1991 to 2006 were obtained from the NYS Department of Health's Statewide Planning and Research Cooperative System. This is a legislatively mandated database that contains hospital discharge data for at least 95% of all acute care hospital admissions in NYS, excluding admissions to psychiatric and federal hospitals. The data included principal diagnoses, hospital admission date, sources of payment, date of birth, sex, race, ethnicity, and street address. About 94% of residential addresses were geocoded to street level, and 5% to zip code level. <1% of the addresses could not geocoded.

2.2. $PM_{2.5}$ concentration and weather data

Fourteen weather regions in NYS that were created so that temperature within each region was relatively homogeneous are shown in Fig. 1 (Chinery and Walker, 2009). Each weather region was assigned a daily average value of temperature and dew point based on hourly meteorologic observations obtained from the National Center for Atmospheric Research (2009) for 18 first-order airport weather stations maintained by the National Weather Service or the Federal Aviation Administration. Daily 24-hour average $PM_{2.5}$ concentration was estimated using all monitoring observations and modeled data for those with missing values as described by Hogrefe et al. (2009).

2.3. Statistical analysis

We conducted time-series analyses to characterize the short-term effect of $PM_{2.5}$ with CVDs and modify it by season and temperature strata using a quasi-Poisson model, while

controlling for possible confounders. We first built the basic model without air pollution, with the following choices regarding the control of confounders:

$$\log E[Y_t] = DOW_t + PUB_t + BOUT_t + ns(temp_t, DF=6)$$
$$+ ns\left(temp_{t,1-3}, DF=6\right) + ns(dewp_t, DF=3)$$
$$+ ns\left(dewp_{t,1-3}, DF=3\right) + ns(t, DF=7/year),$$

where Y_t is the number of CVDs on day t; DOW_t, PUB_t, and $BOUT_t$ are day of week, public holidays, blackout events (8/14/2003 and 8/15/2003); $temp_t$ is the average temperature on day t and $temp_{t,1-3}$ is a moving average of temperature for the previous three days; $dewp_t$ and $dewp_{t,1-3}$ are current day and 3-day moving average of dew point; $ns(\cdot, DF)$ indicates a natural spline with degrees of freedom (DF). The DF numbers used are within the range used in other studies (Cheng and Kan, 2012; Roberts, 2004).

Before examining the results modified by season and temperature, we assessed the short-term effects of $PM_{2.5}$ on the same day (lag 0) and 1, 2, and 3 days before (lag 1–lag 3) on CVDs in 14 NYS weather regions. For this *main model*, we introduced a linear parametric term of $PM_{2.5}$ concentration into the basic model. We also performed this *main* analyses for the five major cause-specific CVD hospitalizations. To examine the season-specific $PM_{2.5}$ effects on CVDs, we extended the *main model* by replacing a linear parametric term of $PM_{2.5}$ concentration with an interaction term between $PM_{2.5}$ and seasons. The four seasons were spring (March to May), summer (June to August), fall (September to November), and winter (December to February).

To investigate whether or not temperature modifies the short-term $PM_{2.5}$ effect on CVDs, we stratified the short-term $PM_{2.5}$ effect by temperature. We defined the low temperature stratum as daily average temperature 25th percentile, middle temperature stratum as daily average temperature between 25th and 75th percentiles, and high temperature stratum as daily average temperature > 75th percentile. The straightforward approach to examine the modifying short-term $PM_{2.5}$ effect by temperature is to replace a linear term of $PM_{2.5}$ concentration with an interaction between $PM_{2.5}$ and temperature strata. We then can utilize these three estimated coefficients and their standard errors to form a statistical test of the $PM_{2.5}$ effect between low vs. middle and high vs. middle temperature strata. However, in this approach, the statistical test is in error since it ignores the correlation in estimation errors of the coefficients being compared. To overcome this problem, we reparameterized the short-term $PM_{2.5}$ effect in the *main model* as the main $PM_{2.5}$ effect at middle temperature stratum and the $PM_{2.5}$ effect differences between temperature strata (middle vs. low and middle vs. high). This *stratified parametric model* provides a simple quantitative comparison of the estimated short-term $PM_{2.5}$ effects of CVDs by allowing $PM_{2.5}$ to have a differential in each temperature stratum and also have a statistical test of the differences between estimated $PM_{2.5}$ effects of temperature strata. This statistical test was generated from the model for coefficients of the $PM_{2.5}$ effect differences between temperature strata. We produce geographic estimates of the association between $PM_{2.5}$ and CVDs based on four $PM_{2.5}$ exposure characterization regions in NYS using Bayesian hierarchical models to combine risk changes across 14 NYS weather regions. Four $PM_{2.5}$ exposure characterization

regions in NYS were created with minimal variation in $PM_{2.5}$ concentration within each region. Details of this development have been previously described by Chinery and Walker (2009). To identify how the weather regions correspond with the $PM_{2.5}$ exposure characterization regions, we overlaid the 14 weather regions onto the four $PM_{2.5}$ exposure characterization regions. All analyses used R 3.0.1 and the splines package for the quasi-Poisson model and the tlnise package for Bayesian hierarchical models (Everson and Morris, 2000).

3. Results

3.1. Descriptive summary

The NYS map describing the 14 weather regions and four $PM_{2.5}$ exposure regions is shown in Fig. 1. Table 1 provides summary statistics for CVDs. Nearly 4.6 million hospitalizations for CVDs took place in NYS over the 16-year period, 71% of which were among NYC, Long Island and Hudson residents. Among overall CVD hospitalizations, 43% were ischemic heart disease and 22% were heart failure. The majority of patients were elderly. Fig. S1a\\S1b (Supplemental materials) provides the distributions of daily average temperature and $PM_{2.5}$ concentration for the whole year across 14 NYS weather regions, 1991–2006. Adirondack & North had the lowest daily average temperature. LGA, JFK and Staten Island had the higher $PM_{2.5}$ concentration.

3.2. Short-term $PM_{2.5}$ effect

Table 2 presents the year-round percent risk changes for daily CVD hospitalization per 10-µg/m^3 increase in $PM_{2.5}$ concentration across four $PM_{2.5}$ exposure regions, 1991–2006. We found evidence of a significant year-round association with a 1.37% increase (95% confidence interval [CI]: 0.90%–1.84%) between $PM_{2.5}$ concentration and CVD hospitalization in NYC, Long Island and Hudson. The short-term $PM_{2.5}$ effect in NYC, Long Island, and Hudson was also significant for cause-specific CVD hospitalization. Its strongest effect occurred on heart failure, followed by hypertension, cardiac dysrhythmias, ischemic heart disease, and cerebrovascular disease.

3.3. Modifications by season and temperature

Table 3 displays the percent risk changes in daily CVD hospitalization per 10-µg/m^3 increase in $PM_{2.5}$ concentration across four seasons in four $PM_{2.5}$ exposure regions, 1991–2006. The seasonal pattern of the short-term $PM_{2.5}$ effects on CVDs is like a monotonic increase from spring to winter except for Mohawk Valley & Binghamton. The strongest effects always occurred in winter and the weakest ones occurred in spring (NYC, Long Island & Hudson, and Central & Western NY) and fall (Mohawk Valley & Binghamton). NYC, Long Island and Hudson had significant short-term $PM_{2.5}$ effects on CVDs ranging from 0.74% to 2.06% in all four seasons; Central & Western NY showed a significant $PM_{2.5}$ effect (1.55%) only in winter. We also estimated year-round 1-day to 3-day lagged CVD morbidity risk associated with $PM_{2.5}$ concentrations. We found little evidence of association with CVD morbidity at these lags (Table S1, Supplemental materials).

Table 4 describes the percent risk changes in daily CVD hospitalization per 10-μg/m³ increase in $PM_{2.5}$ concentration across three levels of temperature in four $PM_{2.5}$ exposure regions, 1991–2006. We found the linear trend of short-term $PM_{2.5}$ effects on CVD hospitalization across three temperature strata except Adirondack & North. The strongest effects occurred at low temperature except for Adirondack & North, and the weakest effects were always found at high temperature in four $PM_{2.5}$ exposure regions. NYC, Long Island & Hudson had significant short-term $PM_{2.5}$ effects at low and middle temperature with 2.20% and 1.63% increased CVDs, respectively. The test results showed the only significant difference in short-term $PM_{2.5}$ effects between middle and high temperature in NYC, Long Island & Hudson.

3.4. Sensitivity analyses

In order to test the sensitivity of the results to the modeling choices described above, we considered several variations of our primary risk model, using different DFs for the smooth function of calendar time and controlling 8-hr maximum average ozone concentration limited to 1000–1800 h, which represents the most likely time for outdoor exposure. Table S2 (Supplemental materials) shows the percent risk change in cardiovascular morbidity per 10 μg/m³ increase in $PM_{2.5}$ using the natural spline of the calendar time with different DFs in the *main* model (5 and 9 df/year). Table S3 (Supplemental Materials) shows the percent risk change in CVDs per 10 μg/m³ increase in $PM_{2.5}$, 1991–2004 with/without controlling ozone. Due to missing values for ozone concentration, we were only able to apply the *main* model with controlling 8-hr maximum average ozone concentration for the sub-dataset (1991–2004). We also investigated the modification of $PM_{2.5}$ effects by temperature using different temperature strata definitions, such as the 33rd/67th and 20th/80th percentiles of the temperature distribution. None of these alternate models produced substantially different CVD morbidity risk estimates.

4. Discussion

The present study used time-series analyses to examine the short-term $PM_{2.5}$ effect and seasonal and temperature modifying effects on CVD hospitalization in NYS from 1991 to 2006.

In our analysis of $PM_{2.5}$ and cardiovascular morbidity, we estimated that cardiovascular morbidity increased 1.37% for a 10-μg/m³ increase in $PM_{2.5}$ in NYC, Long Island & Hudson. Our finding of this significant positive association between $PM_{2.5}$ and CVD hospitalization in NYC, Long Island & Hudson is consistent with previous studies. Dominici et al. (2006) found a 1.28% short-term risk increase in hospitalization for heart failure per 10-μg/m³ increase in same-day $PM_{2.5}$ in 204 US urban counties (population > 200,000) during 1999 to 2002. The underlying biological mechanism for a short-term $PM_{2.5}$ effect may be mediated through autonomic, hemostatic, inflammatory, and vascular endothelial injury/dysfunction (Brook et al., 2010; Zanobetti et al., 2014). For example, $PM_{2.5}$ may trigger the onset of CVD events by increasing blood pressure, heart rate, peripheral vascular resistance, C-reactive protein, oxidative stress, and atherosclerosis, and by decreasing

brachial artery diameter, brachial artery flow, and plaque stability (Fares, 2013; Zanobetti et al., 2014).

We found seasonal variation in associations between cardiovascular morbidity and $PM_{2.5}$ in NYS. Our results identified that the strongest short-term $PM_{2.5}$ effect in CVD hospitalization was in winter, which was consistent with previous findings. Bell et al. (2008) and Bell (2012) reported the highest short-term $PM_{2.5}$ effect (1.49%, 95%CI: 1.09%–1.89%) in winter for respiratory and CVD hospitalizations in 202 US counties, 1999–2005. Fares (2013) reviewed several previous studies of seasonal patterns of CVDs that mostly indicated a peak of CVDs in winter months.

The results of fitting a stratified parametric model suggested that temperature modifies the association between $PM_{2.5}$ and cardiovascular morbidity in NYS. Specifically, we observed stronger $PM_{2.5}$ effects on cardiovascular morbidity for low temperature days. It is consistent with our results of stronger $PM_{2.5}$ effects in winter. The inconsistency between the second strongest effects in summer and the smallest effects on high temperature days could be related to the fact that the temperature range on high temperature days only covered part of the summer temperature range. The summer temperature in NYC, Long Island & Hudson ranged from 10.4 °C to 34.2 °C, but high temperature days had a range from 18 °C to 34.2 °C. The range in summer in NYC, Long Island & Hudson still included some cool days. Previous epidemio-logic studies have reported various findings, including no interactions between air pollution and temperature and higher/lower air pollution effects during summer in morbidity/mortality (Cheng and Kan, 2012; Liu et al., 2013; Zanobetti and Schwartz, 2008; Ren et al., 2006; Ren and Tong, 2006). For example, contrary to our study, Li et al. (2011) have reported that the health effects of ambient air pollutants are enhanced at higher temperature, particularly in regions with typically cooler temperature. Many factors may contribute to this heterogeneity of findings, such as differences in study population characteristics, health endpoints, exposure definition, weather, geographic conditions, climate adaptation, and modeling strategies.

We also observed regional differences in our study; the metropolitan region (NYC, Long Island & Hudson) had significant positive short-term $PM_{2.5}$ effects in most of our analyses, while for the remaining three regions, only Central & Western NY had a significant positive short-term $PM_{2.5}$ effect in winter. Possible reasons are that $PM_{2.5}$ concentration in NYC, Long Island & Hudson was higher than in the other three regions and traffic congestion-related $PM_{2.5}$ in metropolitan regions in summer is higher than in suburban areas. Additional evidence supporting our findings is that other studies evaluating sources of $PM_{2.5}$ found that vehicles contribute up to one-third of observed $PM_{2.5}$ in urban areas (Levy et al., 2010) and urban areas have higher metal levels compared to rural areas (Cibella et al., 2015). Especially, nickel (Ni), which is a component of $PM_{2.5}$, was more related to CVD-related hospitalization than some other components (Ito et al., 2011; Ostro et al., 2009).

Day-to-day air pollution concentrations can fluctuate with changing weather conditions, and temperature is an indicator of change in weather. The relationship between temperature and air pollution is complex and varies seasonally and regionally. The biological mechanisms for $PM_{2.5}$ effects on cardiovascular morbidity modified by temperature are very complicated,

and possible pathways for both temperature and PM$_{2.5}$ are still unclear. However, there are various plausible explanations proposed for our finding of higher PM$_{2.5}$ effects in winter and on low temperature days. There is extensive use of heating appliances in cold seasons and on low temperature days, with coal, oil, diesel, or wood burned and stagnant air masses shaped from low temperature and low wind speed (Fares, 2013). People stay inside with poor ventilation during cold seasons or on low temperature days.

Furthermore, PM$_{2.5}$ total mass alone may be not a sufficient metric in estimating health effects since PM$_{2.5}$ toxicity may vary by its chemical constituents, which vary seasonally and regionally with patterns of source activity and meteorological conditions. There is growing evidence that PM$_{2.5}$ constituents differ in their effects on human health. For example, hospitalization studies have suggested that health effects differ depending upon PM$_{2.5}$ constituents (Peng et al., 2009). Additionally, Bell (2012) reported higher PM$_{2.5}$ effect estimates for CVDs in seasons and areas with higher PM$_{2.5}$ content of Ni, vanadium (V), and elemental carbon. Several studies investigated sources of fine particle composition and oil combustion marked by the presence of Ni and V (Bell et al., 2007; Hains et al., 2007; Lin et al., 2004); PM$_{2.5}$ content of Ni was particularly associated with CVD-related hospitalization (Ito et al., 2011; Ostro et al., 2009). Moreover, previous studies have reported that low temperature activates the sympathetic nervous system and secretion of catecholamine, which resulted in increasing the workload of the cardiopulmonary system; these actions could induce cardiovascular events. For instance, an experimental study showed that frequent cold exposure of rats resulted in development of hypertension (Fares, 2013). These studies suggest that another potential explanation of stronger PM$_{2.5}$ effects in winter and on low temperature days is due to the variation of PM$_{2.5}$ toxicity. Moreover, during the days with high temperature, people tend to stay inside and use air conditioning, thus reducing their indoor and outdoor exposure. Previous studies showed that communities with a higher prevalence of central air conditioning had lower PM$_{2.5}$ effects for CVDs.

There are several major strengths of this study. The stratified parametric model provides parametric estimates and easily compares the difference of parametric estimates by strata. We also repeated our analysis with different DFs for calendar time and adjusted for ozone. The similar findings from these duplicate approaches strengthen the study. Second, we performed statistical tests of differences between PM$_{2.5}$ effect estimates on low vs. middle and middle vs. high temperature, providing a clear indication of whether a significant difference exists. Finally, 16-year time-series data provided an appropriate study time period, and inclusion of 14 NYS weather regions allowed consideration of geographic variation.

This study also has some limitations. We were unable to adjust for lifestyle and other socioeconomic characteristics. For example, we did not know the ventilation status of homes in our study, and we assumed that people kept windows open on high temperature days although this may be modified by the presence of air conditioning. We only examined CVD hospitalization; however, patients with minor symptoms may go to the emergency department.

5. Conclusions

In conclusion, we found a strong short-term effect of $PM_{2.5}$ concentration on CVD hospitalization in NYC, Long Island & Hudson and found the effect to be modified by season and temperature. Specifically, short-term $PM_{2.5}$ effect is stronger in winter and at low temperature days. The implications of these findings are important for policy measures. Since air pollution and temperature have been individually well linked to adverse health outcomes, correct identification of their joint effect provides insight into the health impact from a public health perspective, especially considering the potential effects of climate change.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments/disclaimers

Abbreviations

CI	confidence interval
CVDs	cardiovascular diseases
NYS	New York State
NYC	New York City
PM	particulate matter

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.scitotenv.2016.11.008.

HIGHLIGHTS

- The short-term $PM_{2.5}$ effect on CVDs is identified, with its seasonal pattern and modification by temperature.

- NYC, Long Island & Hudson has year-round $PM_{2.5}$ effect on CVDs.

- The strongest $PM_{2.5}$ effect on CVDs exists in winter and at low temperature days.

- Importance of their joint effect is shown and also provides insight into the health impact from a public health perspective.
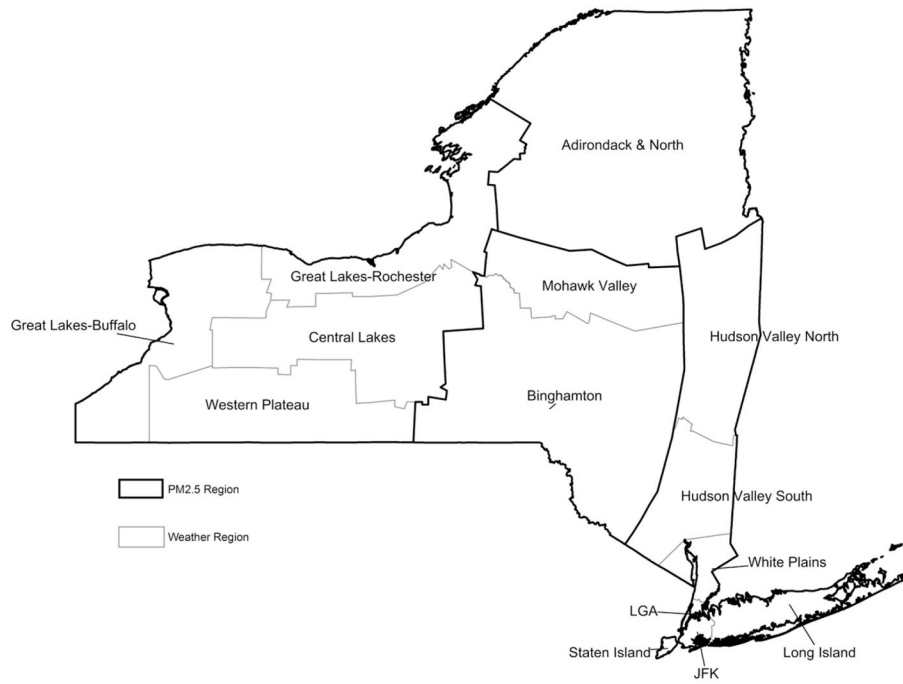
Fig. 1.
14 weather regions and 4 PM2.5 regions.

Table 1

Number and percent of cardiovascular hospitalizations from by patient demographic characteristics, season, and region: New York State, 1991–2006.

	N	Percent (%)[a]
Sex		
Male	2,390,048	52
Female	2,244,562	48
Unknown	140	0
Ethnicity		
Hispanic	299,937	6
Other	3,840,047	83
Unknown	494,766	11
Race		
White	3,288,711	71
Black	630,673	14
Other	462,961	10
Unknown	252,405	5
Age		
0–17	13,533	0
18–44	263,325	6
45–64	1,352,598	29
65–74	1,191,262	26
75+	1,814,032	39
CVD group		
Ischemic heart disease	2,007,862	43
Heart failure	999,264	22
Cerebrovascular disease	672,651	15
Cardiac dysrhythmias	641,614	14
Hypertension	289,011	6
Chronic rheumatic heart disease	243,48	1
Season		
Spring	1,205,343	26
Summer	1,134,091	24
Fall	1,152,013	25
Winter	1,143,303	25
Temperature stratum		
Low	1,170,711	25
Middle	2,340,506	51
High	1,123,533	24
Region		
NYC, Long Island & Hudson	3,277,487	71
Adirondack & North	89,263	2

	N	Percent (%)[a]
Mohawk Valley & Binghamton	324,233	7
Central & Western NYS	943,767	20

[a] due to rounding, the sum of percentage may not be 100.

Table 2

Percent risk change in cardiovascular morbidity per 10 µg/m3 increase in PM2.5 in NYS, 1991–2006% Risk Change (95% CI).

Region	NYC, Long Island & Hudson	Adirondack & North	Mohawk Valley & Binghamton	Central & Western NY
All CVDs	1.37 (0.90, 1.84)[a, b]	−0.43 (−2.33, 1.51)	0.43 (−2.03, 2.95)	0.13 (−0.64, 0.91)
Heart failure	2.16 (1.13, 3.21)[a, b]	−3.54 (−7.55, 0.64)	0.43 (−2.99, 3.98)	0.02 (−1.65, 1.73)
Hypertension	1.93 (0.69, 3.18)[a, b]	6.46 (−1.81, 15.42)	2.03 (−6.05, 10.80)	0.71 (−2.37, 3.88)
Cardiac dysrhythmias	1.52 (0.69, 2.36)[a, b]	0.05 (−4.69, 5.03)	1.43 (−4.34, 7.55)	−0.45 (−3.00, 2.17)
Ischemic heart disease	1.03 (0.49, 1.58)[a, b]	0.53 (−2.27, 3.41)	−0.20 (−2.76, 2.44)	0.28 (−0.83, 1.41)
Cerebrovascular disease	0.83 (0.11, 1.57)[a]	−1.38 (−6.28, 3.77)	0.94 (−3.32, 5.39)	0.28 (−1.35, 1.94)

[a] Significant, p-value <0.05.

[b] Significant, p-value <0.01.

Table 3

Seasonal percent risk change in cardiovascular morbidity per 10 μg/m^3 increase in PM2.5 in NYS, 1991–2006.

Region	% Risk Change (95% CI)			
	Spring[a]	Summer[a]	Fall[a]	Winter[a]
NYC, Long Island & Hudson	0.74 (0.00, 1.49)[b]	1.28 (0.60, 1.96)[b, c]	1.25 (0.56, 1.94)[b, c]	2.06 (1.33, 2.80)[b, c]
Adirondack & North	−1.01 (−4.73, 2.85)	−0.32 (−2.85, 2.27)	−0.44 (−3.55, 2.77)	−0.15 (−4.25, 4.12)
Mohawk Valley & Binghamton	−0.14 (−3.26, 3.08)	0.46 (−2.12, 3.11)	−0.47 (−3.92, 3.11)	1.89 (−1.39, 5.28)
Central & Western NY	−1.12 (−2.65, 0.43)	−0.02 (−1.12, 1.08)	0.10 (−1.06, 1.28)	1.55 (0.17, 2.94)[b]

[a]Spring: 3–5; Summer: 6–8; Fall: 9–11; Winter: 12 and 1–2.

[b]Significant, p-value <0.05.

[c]Significant, p-value <0.01.

Table 4

Percent change in cardiovascular morbidity per 10 µg/m³ increase in $PM_{2.5}$ across three levels of temperature in NYS, 1991–2006.

Region	Low temperature[a]	p-Value[b]	Middle temperature[a]	p-Value[c]	High temperature[a]
NYC, Long Island & Hudson	2.20 (1.34, 3.07)[d, e]	0.12	1.63 (0.93, 2.34)[d, e]	0.04[d]	0.52 (−0.25, 1.29)
Adirondack & North	−0.56 (−4.97, 4.05)	0.62	0.66 (−2.26, 3.68)	0.35	−1.02 (−3.42, 1.45)
Mohawk Valley & Binghamton	1.43 (−2.00, 4.97)	0.67	0.58 (−1.80, 3.02)	0.76	0.06 (−3.25, 3.48)
Central & Western NY	1.35 (−0.03, 2.74)	0.18	0.31 (−0.64, 1.27)	0.25	−0.52 (−1.66, 0.64)

[a]Temperature: °C. Low temperature: daily average temperature 25th percentile; middle temperature: 25th percentile < daily average temperature 75th percentile; high temperature: daily average temperature > 75th percentile.

[b]P-Value for low temperature stratum compared with middle temperature.

[c]P-Value for high temperature stratum compared with middle temperature.

[d]Significant, p-value <0.05.

[e]Significant, p-value <0.01.